## **Amendments to the Claims:**

This listing of claims will replace all prior versions, and listings of claims in the application:

## **Listing of Claims:**

Claim 1 (withdrawn). A method of reducing food consumption in a mammal, said method comprising administering to said mammal a first compound which is a PPAR $\alpha$  agonist and a second compound which is an antagonist of the CB1 cannabinoid receptor, whereby the consumption of food by the animal is reduced.

Claim 2 (withdrawn). The method according to claim 1, wherein the PPARa agonist is an OEA-like agonist.

Claim 3 (withdrawn). The method of claim 1, wherein the PPARa agonist is oleoylethanolamide, palmitoylethanolamide or elaidoylethanolamide.

Claim 4 (withdrawn). The method of claim 1, wherein the antagonist is a pharmaceutically acceptable salt or solvate of a compound of the formula:

$$R_1CH_2$$
 $R_7$ 
 $R_8$ 
 $R_9$ 
 $R_4$ 
 $R_5$ 
 $R_6$ 

wherein  $R_1$  is hydrogen, a fluorine, a hydroxyl, a  $(C_1-C_5)$ alkoxy, a  $(C_1-C_5)$ alkylthio, a hydroxy $(C_1-C_5)$ alkoxy, a group -NR<sub>10</sub>R<sub>11</sub>, a cyano, a  $(C_1-C_5)$ alkylsulfonyl or a  $(C_1-C_5)$ alkylsulfinyl;

 $R_2$  and  $R_3$  are a  $(C_1-C_4)$ alkyl or, together with the nitrogen atom to which they are bonded, form a saturated or unsaturated 5- to 10-membered heterocyclic radical which is unsubstituted or monosubstituted or polysubstituted by a  $(C_1-C_3)$ alkyl or by a  $(C_1-C_3)$ alkoxy;

 $R_4$ ,  $R_5$ ,  $R_6$ ,  $R_7$ ,  $R_8$  and  $R_9$  are each independently hydrogen, a halogen or a trifluoromethyl, and if  $R_1$  is a fluorine,  $R_4$ ,  $R_5$ ,  $R_6$ ,  $R_7$ ,  $R_8$  and/or  $R_9$  can also be a fluoromethyl, with the proviso that at least one of the substituents  $R_4$  or  $R_7$  is other than hydrogen; and

 $R_{10}$  and  $R_{11}$  are each independently hydrogen or a  $(C_1-C_5)$ alkyl, or  $R_{10}$  and  $R_{11}$ , together with the nitrogen atom to which they are bonded, form a heterocyclic radical selected from pyrrolidin-1-yl, piperidin-1-yl, morpholin-4-yl and piperazin-1-yl, which is unsubstituted or substituted by a  $(C_1-C_4)$ alkyl.

Claim 5 (withdrawn). The method of claim 4, wherein said antagonist is of the formula:

or a pharmaceutically acceptable salt thereof.

Claim 6 (withdrawn). A method according to claim 1, wherein the mammal is human.

Claim 7 (withdrawn). A method according to claim 6, wherein said human is overweight or obese.

Claim 8 (withdrawn). A method according to claim 1, wherein the PPARa agonist is a compound of the following formula:

wherein n is any number from 0 to 5;

the sum of a and b can be any number from 0 to 4;

Z is a member selected from  $-C(O)N(R^o)$ -;  $-(R^o)NC(O)$ -; -OC(O)-; -(O)CO-; O;  $NR^o$ ; and S, in which  $R^o$  and  $R^2$  are independently selected from the group consisting of substituted or unsubstituted alkyl, hydrogen, substituted or unsubstituted  $C_1$   $-C_6$  alkyl, substituted or unsubstituted lower  $(C_1$ - $C_6)$  acyl, homoalkyl, and aryl;

up to eight hydrogen atoms of the compound may also be substituted by methyl group or a double bond; and

the molecular bond between carbons c and d may be unsaturated or saturated, or a pharmaceutically acceptable salt thereof.

Claim 9 (withdrawn). A method according to claim 1, wherein said PPAR $\alpha$  agonist is administered with a pharmaceutically acceptable carrier by an oral, rectal, topical, or parenteral route.

Claim 10 (withdrawn). A method according to claim 1, wherein said antagonist is administered with a pharmaceutically acceptable carrier by an oral, rectal, topical, or parenteral route.

Claim 11 (withdrawn). A method according to claim 1, wherein said antagonist and said PPARα agonist are administered together.

Claim 12 (withdrawn). A method according to claim 1, wherein said antagonist and said PPAR $\alpha$  agonist are each administered in an amount below their individual ED<sub>50</sub>.

Claim 13 (withdrawn). A method according to claim 1, wherein said antagonist and said PPAR $\alpha$  agonist are each administered in an amount below their individual ED<sub>10</sub>.

Claim 14 (withdrawn). A method according to claim 1, wherein at least one of said antagonist and said PPAR $\alpha$  agonist is administered in an amount below its ED<sub>10</sub>.

Claim 15 (withdrawn). A method according to claim 1, wherein at least one of said antagonist and said PPAR $\alpha$  agonist is administered in an amount below its ED<sub>50</sub>.

Claim 16 (currently amended). A pharmaceutical composition for reducing food consumption in a mammal, said composition comprising a PPARα agonist and a cannabinoid CB1 receptor <u>antagonist</u>.

Claim 17 (original). The composition according to claim 16, wherein the PPAR $\alpha$  agonist is oleoylethanolamide.

Claim 18 (original). The composition according to claim 17, wherein the antagonist is a pharmaceutically acceptable salt or solvate of a compound of the formula:

$$R_1CH_2$$
 $R_7$ 
 $R_8$ 
 $R_9$ 
 $R_4$ 
 $R_6$ 
 $R_6$ 

wherein  $R_1$  is hydrogen, a fluorine, a hydroxyl, a  $(C_1-C_5)$ alkoxy, a  $(C_1-C_5)$ alkylthio, a hydroxy $(C_1-C_5)$ alkoxy, a group -NR<sub>10</sub>R<sub>11</sub>, a cyano, a  $(C_1-C_5)$ alkylsulfonyl or a  $(C_1-C_5)$ alkylsulfinyl;

 $R_2$  and  $R_3$  are a  $(C_1-C_4)$ alkyl or, together with the nitrogen atom to which they are bonded, form a saturated or unsaturated 5- to 10-membered heterocyclic radical which is unsubstituted or monosubstituted or polysubstituted by a  $(C_1-C_3)$ alkyl or by a  $(C_1-C_3)$ alkoxy;

 $R_4$ ,  $R_5$ ,  $R_6$ ,  $R_7$ ,  $R_8$  and  $R_9$  are each independently hydrogen, a halogen or a trifluoromethyl, and if  $R_1$  is a fluorine,  $R_4$ ,  $R_5$ ,  $R_6$ ,  $R_7$ ,  $R_8$  and/or  $R_9$  can also be a fluoromethyl, with the proviso that at least one of the substituents  $R_4$  or  $R_7$  is other than hydrogen; and

 $R_{10}$  and  $R_{11}$  are each independently hydrogen or a  $(C_1-C_5)$ alkyl, or  $R_{10}$  and  $R_{11}$ , together with the nitrogen atom to which they are bonded, form a heterocyclic radical selected from pyrrolidin-1-yl, piperidin-1-yl, morpholin-4-yl and piperazin-1-yl, which is unsubstituted or substituted by a  $(C_1-C_4)$ alkyl.

Claim 19 (original). The composition according to claim 17, wherein said antagonist is of the formula:

or a pharmaceutically acceptable salt thereof.

Claim 20 (previously presented). The composition according to claim 16, wherein the PPAR $\alpha$  agonist is a fatty acid alkanolamide of the formula:

wherein n is any number from 0 to 5;

the sum of a and b can be any number from 0 to 4;

Z is a member selected from  $-C(O)N(R^o)$ -;  $-(R^o)NC(O)$ -; -OC(O)-; -(O)CO-; O;  $NR^o$ ; and S, in which  $R^o$  and  $R^2$  are independently selected from the group consisting of substituted or unsubstituted alkyl, hydrogen, substituted or unsubstituted  $C_1$   $-C_6$  alkyl, substituted or unsubstituted lower  $(C_1$ - $C_6)$  acyl, homoalkyl, and aryl;

up to eight hydrogen atoms of the compound may also be substituted by methyl group or a double bond; and

the molecular bond between carbons c and d may be unsaturated or saturated.

Claim 21 (original). The composition according to claim 17, wherein said composition is in a formulation suitable for administration by an oral, rectal, topical, or parenteral route of administration.

Claim 22 (original). The composition according to claim 17, wherein said composition is in unit dosage format.

Claims23 and 24 (canceled). position according to claim 22, wherein at least one of said antagonist and said agonist is in an amount below its  $ED_{10}$ .

Claim 25 (currently amended). The composition according to claim 16, wherein the antagonist has an IC<sub>50</sub> for the CB1 cannabinoid receptor <u>as determined in rat cerebellar membrane fractions incubated at 30°C for 90 minutes in 50mM Tris buffer, containing 0.2% bovine serum albumin</u> which is less than one-fourth its IC<sub>50</sub> for the CB2 cannabinoid receptor <u>as determined in rat spleen cells incubated at 4°C for 6 hours in 50mMTris-HBSS buffer containing 0.2% bovine serum albumin.</u>

Claim 26 (original). The composition according to claim 20, wherein  $R^0$  and  $R^2$  are members independently selected from the group comprising hydrogen,  $C_1$ – $C_3$  alkyl, and lower ( $C_1$ - $C_3$ ) acyl.

Claim 27 (original). The composition according to claim 20, wherein a=1 and b=1.

Claim 28 (original). The composition according to claim 20, wherein n = 1.

Claim 29 (previously presented). The composition according to claim 20, wherein  $\mathbb{R}^0$  and  $\mathbb{R}^2$  are each H.

Claim 30 (original). The composition according to claim 20, wherein the bond between carbon c and carbon d is a double bond.

Claim 31 (original). The composition according to claim 20, wherein the alkanolamide or its homologue is according to one of the following formulae:

wherein n is from 1-5 and the sum of a and b is from 0 to 4;  $R^2$  is selected from the group consisting of hydrogen,  $C_1$ - $C_6$  alkyl, and lower ( $C_1$ - $C_6$ ) acyl; and up to four hydrogen atoms of the fatty acid portion and alkanol portion thereof may also be substituted by methyl or a double bond.

Claim 32 (original). A composition of claim 16, wherein the PPAR $\alpha$  agonist is selected from the group consisting of clofibrate; fenofibrate, bezafibrate, gemfibrozil, and ciprofibrate.

Claim 33 (original). A composition of claim 31, wherein the cannabinoid receptor antagonist is rimonabant.

Claim 34 (withdrawn - previously presented). A method of treating an appetency disorder in a human by administering a composition according to claim 16 17.

Claim 35 (withdrawn). A method according to claim 34, wherein the appetite for a food, ethanol, or a psychoactive substance is to be reduced.

Claim 36 (withdrawn - previously presented). A method of claim 34, wherein the  $PPAR\alpha$  agonist is oleoylethanolamide.